Singapore Med J 2000; Vol 41(3):104-106

Ambulatory Blood Pressure Monitoring in Passive Smoking and Atropine – Response in Healthy Volunteers

A Leone, A Guidi, G Bertoncini, A Battaglia

ABSTRACT

Aim: The purpose of this study is to assess the effects of intravenous atropine on blood pressure (BP), systolic BP (SBP) and diastolic BP (DBP), after exposure of healthy volunteers to environmental cigarette smoking.

Methodology: Seventeen non-smoking healthy volunteers (11 men and six women), aged from 20 years to 50 years (mean age: 31.3 ± 8.7), were studied. These subjects received atropine under two conditions: first, in a 60 cubic metres of indoor space unpolluted by cigarette smoke; and second, in the same environment but polluted by 35 ppm carbon monoxide concentration reached by cigarette combustion. BP of each subject was recorded every 15 minutes for two hours by using an Ambulatory Blood Pressure Recorder BR 102.

Results: Mean baseline BPs in a smoke-free environment and in a smoking environment were respectively 120 ± 18 mmHg and 120 ± 25 mmHg for systolic values, and 81 ± 6 mmHg and 84 ± 9 mmHg for diastolic values (P > 0.05). After administration of atropine, mean BPs were respectively 126 (± 24)/80(± 7) mmHg in a smoke-free environment, and 131(± 14)/90(± 2) mmHg after exposure to passive smoking (P > 0.05, no statistical significance).

Conclusion: Our observations showed that BP varied but without statistically significant changes after acute exposure to passive smoking. However, it is known that any increase in BP leads to higher cardiovascular risks.

Keywords: blood pressure, passive smoke, atropine

Department of Medicine
City Hospital Pontremoli
Italy

A Leone, MD
Head

A Guidi, MD

G Bertoncini, MD

A Battaglia, MD

Correspondence to:
Dr A Leone

INTRODUCTION

Earlier studies[1-3] demonstrated the autonomic effects of smoke components on the heart and blood vessels. In the human sinus node, which has been chemically denervated by atropine and propranolol, inhalation of cigarette smoke was associated with increase in heart rate. Passive smoking and atropine caused sympathetic autonomic effects on the heart and blood pressure, although the response of last parameter was not fully clarified . It would seem to increase as shown in our previous study[4] performed under similar experimental methodology. However, the study was mainly conducted to assess cardiac variables such as heart rate and cardiac rhythm rather than the response of blood pressure. Impaired cardiac performance, as shown by heart rate and blood pressure (BP) alterations during exercise stress testing, after acute exposure to passive smoke, has also been described[5-7]. However, controversies seemed to exist on the response of BP – systolic (SBP), or diastolic (DBP) to passive smoke following autonomic stimulation.

The current ambulatory BP monitoring study was conducted to assess the combined effect of passive smoking and atropine, an inhibitor of parasympathetic reflex arcs, on SBP and DBP in non-smoking healthy volunteers.

MATERIALS AND METHODS

Seventeen non-smoking healthy subjects aged from 20 years to 50 years (mean: 31.3 ± 8.7) volunteered for this study. Eleven subjects were men and six women. The characteristics of the studied population are reported on Table I. After providing informed consent, the volunteers underwent a baseline study of SBP and DBP. Each subject then received atropine 1.5 mg iv twice. On the first occasion, the sixty cubic metres indoor space was unpolluted by cigarette

Table I – Characteristics of the studied population

Number	17
Mean age (SD) (yrs)	31.3 (8.7)
Sex	
male	11 (65%)
female	6 (35%)
Mean bodyweight (AD) (kg)	
male	67.4 (5.8)
female	52.3 (7.2)
No previous relevant medical history	

In parentheses: ± SD

smoke, and on the second, the same environment was polluted by 35 ppm carbon monoxide reached by the combustion of 15 – 20 cigarettes within 30 minutes by using a method described elsewhere[6]. In summary, pollution was achieved by cigarette smoking using a switch-over machine connected to a Programmable Infra-red Spectrophotometer (Wilks Mod 80, USA). The switch-over machine consisted of two glass pipes inside with a sucking expelling diaphragm. The first pipe sucks cigarette smoke which is expelled through the other pipe. The Spectrophotometer then measures the environmental carbon monoxide level. Using this device, we could maintain carbon monoxide concentration at the desired level. During the experiment, BP was recorded every 15 minutes for two hours. An Ambulatory Blood Pressure Recorder BR 102 (Schiller, Switzerland) was used to record BP.

STATISTICAL ANALYSIS

All recorded data were calculated as mean and standard deviation (SD). In estimating observed measures and comparing them, 95% confidence interval and both Student's *t*-test and paired *t*-test were used. Statistical significance was determined when a P value less than 0.05 was observed for compared samples.

RESULTS (Tables II and III)

Mean baseline BPs in a smoke-free environment and in a smoking environment were respectively 120 ± 18 mmHg and 120 ± 25 mmHg for systolic values, and 81 ± 6 mmHg and 84 ± 9 mmHg for diastolic values ($P > 0.05$).

After administration of atropine, mean BP were respectively 126 (± 24)/80 (± 7) mmHg in a smoke-free environment, and 131 (± 14)/90 (± 2) mmHg after exposure to passive smoking ($P < 0.05$).

Otherwise, a statistically significant increase in plasma carbon monoxide concentration was detected after 2-hour stay in passive smoke (0.8 ± 0.2 vs 2.1 ± 1.9, $P < 0.01$).

Table II – SBP-mean (SD), mmHg- of the studied subjects in a smoke-free and smoking environment before and after atropine

	Smoke-free env.	Smoking env.	*t*-test (smoke-free/ smoking)
Baseline SBP	120 (18)	120 (25)	p = NS (.15)
Atropine SBP	126 (24)	131 (14)	p = NS (.13)
t-test (baseline/atropine)	p = NS (.09)	p = NS (.13)	
SBP 95% confidence interval:	Smoke-free/	111 → 129	
	Smoking env.	107.5 → 132.5	

Table III – DBP-mean (SD), mmHg- of the studied subjects in a smoke-free and smoking environment before and after atropine

	Smoke-free env.	Smoking env.	*t*-test (smoke-free/ smoking)
Baseline DBP	81 (6)	84 (9)	p = NS (.05)
Atropine DBP	80 (7)	90 (2)	p = NS (.04)
t-test (baseline/atropine)	p = NS (.04)	p = NS (.04)	
SBP 95% confidence interval:	Smoke-free/	79.5 → 82.5	
	Smoking env.	79.5 → 88.5	

DISCUSSION

The relationship between smoking and BP has not been fully clarified in spite of several studies on the subject[1-3,8-10]. These studies have appeared to show an involvement of the autonomic system that influences some cardiovascular parameters, particularly heart rate, cardiac rhythm and BP. Chemical denervation of human sinus node by atropine and /or propranolol was characterised by increase or decrease in heart rate not always accompanied by similar changes in cardiac rhythm or blood pressure.

We could find only one study[4] that had used ambulatory monitoring to quantify cardiac variables after exposure to passive smoke in subjects pre-treated with atropine. This study evaluated the effects of passive smoking plus atropine on heart rate of healthy volunteers and athletes, and found that different responses in heart rate characterised the two groups. In the smoking environment, an atropine-induced increase in heart rate was seen among athletes, whereas a smoking-induced increase in heart rate was observed for the healthy non-athletes. The findings on BP in this study were only preliminary observations. Reported data appeared to show that exposure to passive smoke in volunteers pre-treated with atropine did not modify BP statistically.

In the present study, only parasympathetic arcs were blocked by atropine so that increased stimulation from sympathetic ganglia could be observed. Therefore, one or more of the components of passive smoke could exacerbate sympathetic but not parasympathetic reflexes.

It is generally agreed that nicotine causes changes in cardiovascular variables[11,12] through stimulation of sympathetic ganglia. However, one study[3] appeared to show a direct effect of cigarette smoke on some (chemically denervated) cardiovascular structures.

Our observations suggested that there was a non-statistically significant increase in both SBP and DBP during exposure to passive smoke in subjects pre-treated with atropine. However, it is uncertain if any increase in BP may lead to a higher rate of subsequent cardiovascular events.

Moreover, atropine administered at a dose effective to block transiently parasympathetic arcs (1.5 mg iv), did not increase significantly BP measures compared to the baseline measures in the volunteers of this study.

CONCLUSION

In the present study, there is a trend towards increase in BP after acute exposure to passive smoking in healthy subjects pre-treated with atropine.

Although a mild rise in BP may heighten cardiovascular risk, further studies are needed to determine if the elevation of BP during exposure to cigarette smoke is clinically significant.

REFERENCES

1. Westfall T, Cipolloni P, Edmundowicz A. Influence of propranolol on hemodynamic changes and plasma catecholamine levels following cigarette smoking and nicotine. Proc Soc Exp Biol Med 1966; 123:174-9.
2. White S, McRitchie R. Nasopharingeal reflexes: integrative analysis of evoked respiratory and cardiovascular effects. Aust J Exp Biol Med Sci 1973; 51:17.
3. Volosin KJ, Brachfeld C, Beauregard LM, et al. Effect of cigarette smoke on sinus node automaticity. Am J Cardiol 1990; 65:243-5.
4. Leone A, Battaglia A, Fabiano P, et al. Effects of passive smoking and atropine on cardiovascular variables in healthy volunteers and athletes. J Smoking-Related Dis 1995; 6:43-7.
5. McMurray RG, Hicks LL, Thompson DL. The effects of passive inhalation of cigarette smoke on exercise performance. Eur J Appl Physiol 1985; 54:196-200.
6. Leone A, Mori L, Bertanelli F, et al. Indoor passive smoking: its effect on cardiac performance. Int J Cardiol 1991; 33:247-52.
7. Mori L, Bertanelli F, Fabiano P, et al. Indoor passive smoking and cardiac performance: mechanisms able to cause heart failure. J Smoking-Related Dis 1993; 4:213-7.
8. Cellina GU, Honour AJ, Litter WA. Direct arterial pressure, heart rate, and electrocardiogram during cigarette smoking in unrestricted patients. Am Heart J 1975; 89:18-25.
9. Roth GM, McDonald JB, Sheard C. Effect of smoking cigarettes and of intravenous administration of nicotine on electrocardiogram, basal metabolic rate, cutaneous temperature, blood pressure and pulse rate of normal persons. JAMA 1944; 125:761-8.
10. Asano M, Ohkubo C, Sasaki A, et al. Some physiological responses to acute passive smoking in healthy young adults. Tokai J Exp Clin Med 1985; 10:355-62.
11. US Department of Health and Human Services. The health consequences of smoking: cardiovascular disease. A report of the Surgeon General. Washington, DC 1983; DHSS publication no. (PHS) 84-50204.
12. Leone A. Il fumo di sigaretta: un potente fattore di rischio cardiovascolare. Il Cuore 1991; 5:573-80.